Plasma Surface Activity in Atherosclerosis

SIR,—Your leading article (12 December, p. 1477) implies that surface-active agents may be of therapeutic value in atherosclerosis. It is pertinent to inquire of evidence that impairment in plasma surface activity is implicated in the aetiology of this disease and its complications.

The first known demonstration of such a relationship was in 1958. It led to a new theory of atherogenesis¹⁻⁶ which embraces other existing theories and integrates evidence relating atherosclerosis and ischaemic heart disease to fibrin deposition, a raised serum-cholesterol level, platelet aggregation, and thrombus formation. The evidence suggested that impairment in plasma surface activity is of greater importance than a raised serum-cholesterol level. It follows that attempts to reduce serum cholesterol should only be made if the clinician ensures there is no accompanying deleterious effect on plasma surface activity. Unfortunately this is not being done despite the existence for six years of a simple laboratory method of determination, discussed in your columns previously.^{5, 6}

Impaired plasma surface activity may indeed be the key to the understanding of the mass of clinical and experimental evidence which otherwise appears to suggest a diverse aetiology. The many factors involved begin to assume a pattern. If this is so, and I know of nothing to refute it, then surely, as a matter of urgency, research efforts should be co-ordinated to determine what are the chemical factors leading to reduced plasma surface activity. This may be the road leading to logical therapy.—I am, etc.,

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- ³ ——— *Clin. Sci.*, 1959, 18, 263.
- ⁴ ——— and Clark, A., *ibid.*, 1961, 20, 279.
- ⁵ *Brit. med. J.*, 1962, 2, 108.
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Natural History of Atherosclerosis

SIR,—The paper by Drs. C. J. Schwartz and T. A. White (5 December, p. 1415) is a valuable contribution to our knowledge of the natural history of human atherosclerosis. It shows once more that even advanced mural arterial lesions do not necessarily lead to gross ischaemic damage of vital organs. Schwartz and White emphasize that in the younger age groups stenosing lesions of the renal arteries are much more common in males than in females, but that this sex difference is abolished or reversed in the older age groups. They stress the marked predominance of a severe grade of intra- and extra-renal arterial lesions in women aged over 65.

These necropsy findings on renal arteries are in complete agreement with my radiological observations on the incidence and severity of calcified atherosclerosis of the abdominal aorta in males and females over 50 years of age.¹ They revealed that in the 50–60 age group the incidence of calcified atherosclerosis is greater in males than in females. This sex ratio is reversed in the higher age groups, when the incidence—and, in particular, the severity—of calcified atherosclerosis in

women far exceeds that in males. It was pointed out that the presence of calcified atherosclerosis in the abdominal aorta, as seen on radiographs, is usually a reflection of more widespread atherosclerosis.

These observations may, at least partly, explain why Schwartz and White found a striking sex difference in the incidence and severity of atherosclerotic lesions in the renal arteries in the higher age groups.

There is considerable evidence indicating that the loss of the anti-atherogenic effect of ovarian function, caused by physiological or artificial menopause, leads to a rapid advance in the incidence and severity of atherosclerosis. This is in contrast to males, in whom atherosclerosis starts at an earlier age and progresses more slowly with age.

From these and other observations an attempt can be made to construct a pattern of the natural history of human atherosclerosis and its complications. Localized stretches of calcified lesions, causing narrowing of the ostia of otherwise unaffected arteries, tend to lead more often to serious complications than the diffuse atherosclerotic lesions. The former are frequently seen in the younger and middle-aged individuals, predominantly male, while the latter are commonly present in elderly women and do not prevent them from often reaching a ripe old age. It should, however, be pointed out that in the long run even the clinically silent

lesions carry a high risk, and they have been aptly compared to the underwater portion of the iceberg.² This is shown by the observation that the rate of increase in the complications of atherosclerosis is greater in females than in males in the seventh and eighth decades of life.—I am, etc.,

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Atherosclerosis and Carcinoma

SIR,—In your leading article (12 December, p. 1477) "Is Atherosclerosis Reversible?" you mention necropsies on malnourished concentration-camp victims, but not those on malnourished victims of chronic disease, such as carcinoma. I have sometimes wondered, in noting only a minor degree of atherosclerosis of the large arteries in a proportion of such necropsies, why this heretical idea has not yet reached one of our modern Councils of Nicaea.—I am, etc.,

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Acute Hemiplegia in Childhood

SIR,—Dr. E. R. Bickerstaff (11 July, p. 82) in his paper on acute hemiplegia in childhood makes the interesting postulate that the pathology of this syndrome may be an inflammatory carotid arteritis with intimal damage and peripheral embolization, resulting in cerebral lesions ranging from transient ischaemia to frank infarction. He considers that the pathogenesis of the carotid arteritis may be previous throat or cervical-node infection or trauma.

I would like to record the case history of a patient who was recently under our care with similar clinical and radiographic findings.

A.B., a previously healthy 12-year-old white female, was admitted to Groote Schuur Hospital with a hemiplegia of acute onset. The clinical history revealed previous apparently minimal trauma to the right side of the neck as a result of a fall from a horse three weeks prior to admission to hospital. She had had a mild throat infection a week before admission. Her present illness began while again out riding when she suddenly complained of intense pain in the right eye and felt faint. She was confused and found to be unsteady on her feet. A few hours later she complained of severe right-sided headache, and vomited once. The following morning she was noted to have a left-sided hemiplegia and dysphasia.

On examination in hospital she was found to be drowsy with a left facial lower-motor-neurone paralysis and flaccid left hemiplegia

with a left extensor plantar response. A small scar was noted on the right side of the neck overlying the common carotid artery.

After 24 hours she was fully conscious and power had begun to return to the left leg. By the fourth day she could move her leg well and considerable movement had returned to the left